

## Comment on “Parasites as a Viability Cost of Sexual Selection in Natural Populations of Mammals”

We appreciate the study by Moore and Wilson (1) that investigated the relation between sex-biased parasitism (SBP) and sex-biased mortality (SBM) in mammals. Regrettably, however, Moore and Wilson ignored the association between SBP and a crucial factor in parasite exposure: home range. Sex-based differences in home range are extremely common in mammalian species (2). Logically, mammals that traverse a greater area are exposed to a greater number of parasites because they interact with more parasite habitat and with more individuals carrying infectious parasites. Of course, body size is correlated with home range, because larger mammals can typically defend larger territories and because maintaining a greater body size often requires foraging over a greater area (3, 4).

We therefore argue that sexual difference in home range, rather than body size, is a more proximate (yet unappreciated) mechanistic basis for SBP. For example, a doubling in body size is likely to yield much less additional parasitic exposure compared to a doubling in home range. The impact of increased exposure to parasite habitat would presumably be most evident for free-living parasites, which may explain why Moore and Wilson found the largest effect size of SBP

for arthropod parasites but a nonsignificant effect size for unicellular parasites. Although Moore and Wilson also used a mating system variable (monogamous or polygynous) to show an association between SBP and sexual selection, they did not consider that polygynous males typically frequent much larger areas relative to females (5). To secure mates, polygynous males must thus increase their probability of traversing parasite habitat, while interaction with each additional mate represents another potential source of infection.

Moore and Wilson (1) suggested that their findings are consistent with male-biased mortality as a result of males investing in enhanced growth and differential resource allocation at the expense of their immune systems. In a related Perspective article, Owens (6) used this logic to explain part of human SBP with statistics from the World Health Organization (7). If males were truly more susceptible to parasitic and infectious disease due to higher energetic or hormonal investments in growth and function (rather than behavioral differences), then we would expect a high incidence of disease mortality during the intensive human development before age 25. Although this is obviously not the case [see

corrected figure from Owens (8)], we question the validity of using the underlying data set to investigate this question in the first place: 48% of the mortality due to parasitic and infectious disease in these data is from AIDS, an affliction strongly influenced by the behavior of adult males.

Our new perspective on SBM changes little for insurance companies—males still “out-die” females. However, it does indicate that public-health programs can reduce mortality with behavioral interventions, rather than assuming that males are doomed to immune system inferiority. We find this to be good news for males.

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